

SCIENTIFIC LETTER

Dual chamber pacing in patients with severe heart failure on β blocker and amiodarone treatment: preliminary results of a randomised study

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Standard dual chamber pacing was first used as adjunctive treatment for severe congestive heart failure in the early 1990s.¹ It was proposed that a short atrioventricular delay reduced presystolic mitral regurgitation.^{2–3} It also may correct chronotropic incompetence and protect against fatal bradyarrhythmias.

We performed a randomised study to test whether DDDR pacing with optimised atrioventricular (AV) delay and reversal of drug induced bradycardia by rate responsive pacing was beneficial in heart failure patients who were receiving combined treatment with amiodarone and β blockers as empirical prophylaxis against sudden tachyarrhythmic death.⁴

In 82 patients with severe heart failure submitted for heart transplantation and without a conventional pacemaker indication between 1996 and 1998 (85% male; 52% idiopathic dilated cardiomyopathy), treatment with low dose amiodarone (1000 mg/week) plus titrated doses of carvedilol (target 50 mg/day) was instituted. In addition, a dual chamber pacemaker was installed with a minute ventilation sensor (Chorus RM).

After a three month wash out period (no pacing), the devices were randomly programmed in VVI backup mode with a basal rate of 40 impulses/min and a hysteresis of 25% ($n = 41$, “no pacing”), or in DDDR mode with heart rates from 65 to 120 beats/min and haemodynamically optimised AV delay ($n = 41$). The two groups (VVI, DDDR) showed similar baseline values in New York Heart Association functional class (mean (SD): 3.24 (0.6) v 3.18 (0.7)), heart rate (88 (14) v 88 (16) beats/min), and ejection fraction (24.8 (9)% v 24.4 (11)%) and were observed for 2.8 (1.1) years.

In contrast to VVI inhibition mode, DDDR pacing shortened the PR interval from 217 (55) to 172 (19) ms ($p < 0.001$) but prolonged the QRS interval from 143 (38) to 174 (30) ms ($p < 0.001$). The mean resting and 24 hour heart rates after one year were 60 (8) and 65 (8) beats/min in the VVI group, and 69 (9) and 74 (8) beats/min in the DDDR group ($p < 0.01$ between the groups). In the VVI patients (without pacing), the left ventricular ejection fraction increased more than in the DDDR patients after one year (40 (12)% v 32 (11)%, $p = 0.04$ between the groups). Changes in left ventricular ejection fraction (LVEF) were negatively correlated with heart rate changes ($r = -0.47$, $p < 0.001$). In other words, heart rate lowering seemed to be closely associated with an increase in LVEF. This may partly be explained by the fact that when cardiac output and volumes were unchanged, the ejection fraction has to be increased. Also, left ventricular end diastolic diameter was slightly higher and left ventricular stroke work significantly lower in paced patients. There was no effect on mitral regurgitation.

In contrast to our hypothesis, there were more cardiac events such as death, the need for heart transplantation, or the need for upgrade to biventricular pacing in the DDDR group than in the VVI group (17 v 8, $p = 0.03$, table 1). On the other

Table 1 Cardiac events during the observation period

	VVI	DDDR	p Value
n	41	41	
Observation time (years) (mean (SD))	2.8 (1.1)	2.7 (1.1)	NS
Heart transplantation	2	5	
CHF death	1	3	
Sudden death	4	6	
Upgrade to biventricular pacing because of refractory heart failure	1	3	
Total number of major cardiac events	8 (19%)	17 (41%)	0.03

CHF, congestive heart failure.

hand nine of the 41 patients in inhibited VVI mode (21%) had to be reprogrammed to DDDR mode because of symptomatic bradycardia (crossover).

COMMENT

We conclude that conventional DDDR pacing with optimised AV delay is not superior to VVI pacing and may be deleterious in patients with severe heart failure treated with β blockers and amiodarone, and who do not have a conventional indication for pacing. In particular, no effect on mitral regurgitation could be observed. These data contrast with the findings in patients undergoing biventricular pacing.⁵

Two mechanisms may be responsible for our negative result: first, the increased, possibly inappropriate, heart rate induced by the minute ventilation sensor may have impaired ventricular filling; second, the direct negative effects of right ventricular pacing in the DDDR group may have induced ventricular dyssynchrony. Our findings raise the possibility that heart failure patients may improve steadily over at least one year without continuous pacing. Backup pacing was necessary in a substantial number of patients treated with a combination of amiodarone and β blockers, and so patients on such treatment should be monitored closely. Pacing may be an adjunct that can maximise medical heart failure treatment, obviating the need to withdraw negative chronotropic drugs.

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IMAGES IN CARDIOLOGY.....

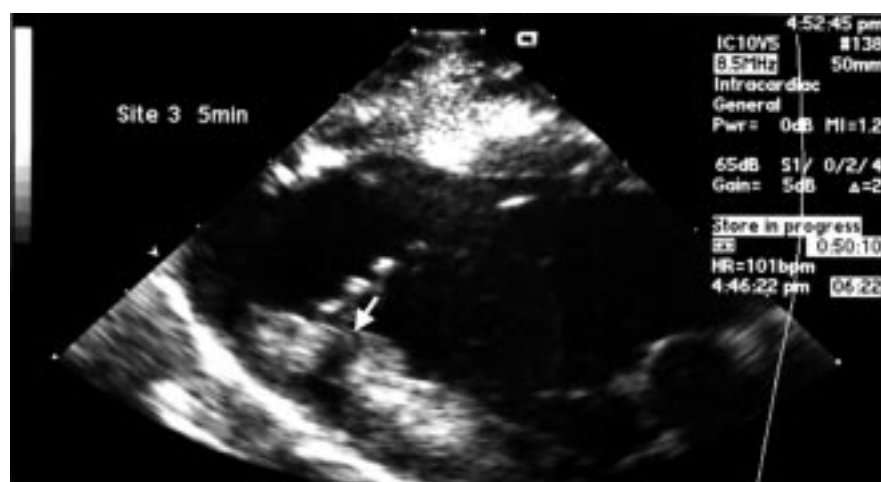
Histological gap between ablation lesions detected by phased array intracardiac echocardiography

Discontinuous linear ablation lesions can be a cause of proarrhythmias such as re-entrant tachycardias. Assessment of the wall contact and prevention of proarrhythmic gaps remain crucial. The technique of direct visualisation of the discontinuities between ablation lesions is not well established. Intracardiac echocardiography (ICE) is a technique that can be used to visualise various intracardiac structures that are not visualised on fluoroscopy and which allows precise localisation of intracardiac catheters relative to these anatomical structures. A deflectable 8.5 MHz phased array ICE catheter (Acu Nav, Acuson Inc) detected a real time histological gap on the posterior wall of the left ventricle in vivo in a canine model (below left, arrowhead). Low power energy deliveries were made in the left ventricle for 60 seconds. The target temperature was 60° with a maximum power of 40 W, which was the same as that used in the

clinical setting. The ablation lesions were vividly demonstrated by the presence of an increase in the tissue density. The gap between the two ablation lesions was detected as a relatively hypoechoic area. The real time measurement of the maximum distance between the two lesions was 3.1 mm. A histological examination revealed sharply demarcated ablation lesions and a histological gap (below right). Macroscopically, the maximum size of the gap measurement was 3 mm. The size of the gap in ICE may be useful in the direct identification of discontinuation lesions in linear low power ablation.

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Post-infarction ventricular septal defect with aneurysm

A 65 year old woman was transferred to our hospital eight days after sustaining an inferior myocardial infarction. She had undergone thrombolysis and was discharged after an uneventful early course of recovery. She was readmitted two days later in cardiac failure, with a new systolic murmur. Two dimensional echocardiography in our unit demonstrated a large defect in the basal muscular ventricular septum with an aneurysmal portion of intact tissue bulging into the right ventricle. Colour Doppler examination showed left-to-right flow through the defect, reaching the right ventricle via an opening in the basal end of the aneurysm. The right ventricle was dilated and there was a moderate pericardial effusion. The patient was brought to theatre where it was found that virtually the

whole of the ventricular septum had infarcted. Multiple attempts were made to repair the defect, but the necrotic nature of the surrounding tissue made adequate repair impossible. The patient died shortly after the operation.

The microarchitecture of the septum, with a laminar arrangement of myocytes and connective tissue, leads to the existence of possible cleavage planes. Conditions of infarction combined with the hydrostatic pressure of the left ventricle can cause splits within the wall, giving rise to the unusual appearances seen here.

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